Ruwen Jou,* Pei-Chun Chuang,* Ying-Shun Wu,† Jing-Jou Yan,‡ and Kwen-Tay Luh§

*Center for Disease Control, Taipei, Taiwan, Republic of China; †Chest Hospital, Tainan, Taiwan, Republic of China; ‡National Cheng Kung University Hospital, Tainan, Taiwan, Republic of China; and §National Association of Tuberculosis, Taipei, Taiwan, Republic of China

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Address for correspondence: Ruwen Jou, Reference Laboratory of Mycobacteriology, Center for Research and Diagnostics, Center for Disease Control, Department of Health, 161 Tun-Yang Street, Nan-Kang, Taipei, 115, Taiwan, Republic of China; email: rwj@cdc.gov.tw

Enrofloxacin in Poultry and Human Health

To the Editor: Following logic similar to that recently used by the US Food and Drug Administration to withdraw approval for enrofloxacin, a recent letter estimated that fluoroquinolone use in poultry could compromise responses to antimicrobial drugs in >24,000 persons per year in the United States (1). However, >99.9% of this estimated risk appears to result from incorrect assumptions. Potentially important corrections include the following: 1) not attributing resistance from foreign travel and human ciprofloxacin use to domestic use of enrofloxacin in poultry (this could reduce the estimated risk by 1/3) (2); 2) updating the estimated fraction of human foodborne Campylobacter infections caused by poultry to reflect declines in microbial loads on chicken carcasses since 1992 reduces the estimated risk by a factor of perhaps 1/10 (3) (the cited 90% estimate by Hurd et al. [1] was intended for use as part of a conservative upper-bounding analysis, not as a realistic point estimate); 3) replacing an assumption that 10% of infected persons would benefit from antimicrobial drug treatment (rather than, for example, erythromycin) by a more realistic value of 0.6% (4) would reduce the estimated risk by a factor of 0.6/10 = 0.06; 4) replacing an assumption that fluoroquinolones are prescribed for all affected patients receiving antimicrobial drug treatment (whether treated in hospital) would reduce the estimated risk by a factor of 1/6 (5); and 6) recognizing that reducing enrofloxacin use may not decrease fluoroquinolone resistance in all Campylobacter spp. from food animals (effect not quantified) (6). Together, such changes reduce the estimated risk by a factor of at least (1/3) × (1/10) × (0.6/10) × (1/2) × (1/6) = 0.00017, or by >99.9%.

More notably, the calculation in (1) also wrongly assumes that the fraction of patients with fluoroquinolone-resistant infections times the fraction of infections caused by poultry gives the fraction of patients with compromised response caused by fluoroquinolone use in poultry. As a simple counterexample, suppose that 80% of all infections were caused by poultry, with the rest caused by something else (e.g., water), and that all and only the 20% of infections caused by the latter source are resistant. Then the procedure in (1) would estimate (80% of infections caused by poultry) × (20% of infections resistant) = 16% as the fraction of resistant infections caused by poultry, even though the correct answer is zero. Thus, the basic logic of the calculation is flawed.

Louis Anthony Cox, Jr*†
*Cox Associates, Denver, Colorado, USA; and †University of Colorado Health Sciences Center, Denver, Colorado, USA

References

Cox also misquotes Busby et al. (3) when he asserts that only 0.6% of persons with Campylobacter infections benefit from antimicrobial drugs. The Busby article states that 0.6% of persons with Campylobacter infections need “hospitalization,” not how many would benefit from antimicrobial drug therapy. Cox has thus made a misleading attribution (something he has previously been found to do [5]).

Busby et al. (3) estimated that in 1993, ≈1,500,000 persons in the United States acquired Campylobacter infections from food sources. Even if the proportion who can benefit from receiving antimicrobial drugs is as low as 2%, this translates to 30,000 persons. If 20% of these infections were caused by fluoroquinolone-resistant Campylobacter spp., then 6,000 persons would potentially have their therapy and outcome compromised, rather than the 1 person that Cox would have us believe. More realistic is the figure of 24,000 persons estimated previously to be at risk of having an adverse outcome (or =285 persons for every 1 million chickens treated with fluoroquinolones) (1). Cox’s assumptions and calculations thus seem flawed and unrealistic.

Peter Collignon*
*The Canberra Hospital, Woden, Australian Capital Territory, Australia

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